

on the artery proximal and distal to the sheath (Fig. 1, A). The sheath is removed under direct vision and the arterial defect is closed with three or four interrupted 6-0 polypropylene sutures. The sutures are placed perpendicular to the vessel including all arterial layers in the repair. Alternatively, the artery is dissected only minimally on its anterior wall and pinched on either side of the sheath insertion site with atraumatic forceps to control bleeding while sutures are placed to approximate the two sides of the laceration (Fig. 1, B).

After closure of the arterial defect, careful wound hemostasis is obtained with cautery, and it is closed in layers. Heparin infusion may be safely continued during and after this procedure, until adequate long-term anticoagulation with warfarin is instituted. In a group of patients so treated, no significant bleeding or hematoma formation occurred.

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#### REFERENCES

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2. Gardiner GA, Harrington DP, Koltun WK, et al. Salvage of occluded arterial bypass grafts by means of thrombolysis. *J VASC SURG* 1989;9:426-31.

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#### Hemodynamic basis of stasis ulceration – A hypothesis

*To the Editors:*

We read with interest the paper by Raju and Fredericks<sup>1</sup> in which they introduce a new concept of Valsalva-induced venous hypertension that may explain those cases with stasis ulceration and normal ambulatory venous pressure.

We also have found cases of venous ulceration that are not explained by ambulatory venous hypertension and therefore have started introducing Valsalva-induced foot venous pressure testing into our venous pressure assessment program.

It has soon become apparent, however, that many patients are unable to sustain a Valsalva effort of 30 to 40 mm Hg for sufficient time to reach a uniform plateau in the pressure tracing, thereby immediately introducing a measure of pulmonary reserve into the test.

We would suggest that all readings should be taken a given time after the commencement of the Valsalva effort (e.g. 5 seconds) to compensate for this. Unfortunately, it is

not clear from the paper cited whether the Valsalva effort was standardized in such a manner. Elucidation of this point would be useful to those considering introducing this test into the repertoire of their vascular studies unit.

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#### REFERENCE

1. Raju S, Fredericks R. Hemodynamic basis of stasis ulceration – a hypothesis. *J VASC SURG* 1991;13:491-5.

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#### Reply

*To the Editors:*

We have tried several variations of the Valsalva test in our patients. Initially maximal Valsalva effort was used without actually measuring the strength of the effort. A 5-second delay as suggested by Drs. Payne and Barrie was in fact tried in a series of patients because we had the same concerns as those expressed by them.

In the last several years a graduated Valsalva technique whereby the patient blows into a manometer to exert a 30 to 40 mm Hg pressure has been used to standardize the technique. It is useful for the technician to monitor the effort by the patient by placing a hand lightly on the abdomen. In practical terms, we have found little difference in the foot venous pressure elevation irrespective of the technical variation used.

It has been our experience that the foot venous pressure plateaus quite early during the Valsalva effort thus minimizing variability from differences in the effort itself. The test is highly reproducible.

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#### Comments on the diagnosis of subclavian (or innominate) artery steal syndrome

*To the Editors:*

The purpose of this brief communication is to substantiate the statement that the term "steal" misrepresents the actual hemodynamic changes.

As shown in Fig. 1, the transient decrease in upper